Chapter 8

EXERTIONAL HEATSTROKE IN THE ISRAELI DEFENCE FORCES

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INTRODUCTION

Most victims of exertional heatstroke (EHS) are highly motivated, healthy, young individuals who exert themselves beyond their physiological capacity (Exhibit 8-1). In 1989, Sir Roger Bannister commented on EHS in the armed forces in a letter to *The Times* of London:

> The notion that courage and esprit de corps can somehow defeat the principles of physiology is not only wrong but dangerously wrong; life can unnecessarily be lost.[1]

Very often military personnel, especially in their basic training regimen, exert themselves to the edge of their physiological ability. We in the Israeli Defence Forces (IDF) also intentionally encourage motivation and push the soldiers to perform to the edge of their ability, with very limited safety margins. Although motivation is very important in "courage and esprit de corps," it might become a noxious agent. Therefore, commanding officers should be aware of their subordinates' ability, monitor them during training, identify potential risk factors for heatstroke, and watch that their subordinates do not exert themselves beyond their capacity.

In a review of 82 cases of EHS that occurred in the IDF during the years 1988 through 1996,[2] most cases were found to have occurred during basic training (57%); an additional 21% occurred during screening tests for special forces—in which motivation is a key issue. Most of the cases of EHS occurred in highly motivated and relatively unfit soldiers during short marches lasting less than 2 hours (46% of all cases that occurred during marches) or during short distance (< 5 km) runs (57% of all the cases that occurred during runs).

EXHIBIT 8-1

EXERTIONAL HEATSTROKE: THE ILLNESS OF THE OVERMOTIVATED

A squadron of highly motivated special forces soldiers was engaged in an intense physical training regimen under desert conditions; the temperature of the ambient air was 40°C to 45°C. Two of the soldiers, who had been absent for several weeks from regular training with the squadron, joined their friends only on the fourth day of the maneuver. At the end of this day, these soldiers suffered from dizziness, headaches, disorientation, nausea, and confusion; their friends described their behavior as "irrational and strange." They were given fluids (intravenous infusion of Hartman's solution) but no measurements of body temperature were taken.

After a 4-hour night rest, and without seeking medical advice, the soldiers continued with regular training, which was carried out in very hot climatic conditions and in difficult terrain. At midday both soldiers collapsed within 15 minutes of each other. Their heart rates were high, they vomited and were tachypneic, but body temperature was not measured. The medic made a diagnosis of "heatstroke," which was later confirmed in the emergency department and at the postmortem examination.

A PREVENTABLE CONDITION

The Israeli experience indicates that prompt recognition, attention, and treatment usually result in complete recovery from EHS, and that EHS can be prevented by following simple regulations and proper health education.

Exertional heat illness is a sporadic phenomenon. It follows that an individual's tolerance to heat is compromised by some underlying factors, many of which have been identified in the literature.[3] However, based on military experience in the young active population, four factors are of major importance in the development of EHS: low physical fitness, improper acclimation to heat, acute febrile illness, and hypohydration.[2] Although these factors relate to the individual, they can be identified by commanders and thus be controlled.

Preventive Measures

The understanding that human ability has its limits led to the issuing of simple regulations that proved to be helpful in reducing the number of heat casualties.[4] Cumulative experience suggests that

- physical efforts should match the individual's capacity,
- physical activity should be limited under severe heat load,
- rest periods should be scheduled during activity, and
- adequate hydration should be emphasized.

These measures are effective if they are targeted to the individual as well as to the organization. The mainstay is education.

Medical personnel who supervise events that involve strenuous activity must, prior to the exercise, examine, evaluate, and when necessary, eliminate a subject who is predisposed to heatstroke, or who, during the exercise, exhibits prodromal signs of heatstroke (discussed below). Medical personnel should also have the authority to cancel maneuvers and other strenuous activities when weather conditions are adverse. These simple measures, which were implemented in the IDF and have been in practice for many years, are also the official position of the American College of Sports Medicine.

In the IDF, each case of EHS is subjected to an inquest by an investigation board, whose members are nominated ad hoc. Its primary aim is to examine the circumstances of the case in detail and to draw the necessary conclusions. If the inquest finds that regulations have been disregarded, commanders are considered responsible, and disciplinary measures—which may include those resulting from courts martial—are taken.

The Heat Tolerance Test

Soldiers who have recovered from EHS undergo a standard heat tolerance test (HTT) 6 to 8 weeks after the injury to screen for congenital or acquired factors that might compromise body temperature regulation.[3] The test consists of walking on a treadmill at 3.1 mph (~ 5 km/h), 2% elevation, for 2 hours, in a climatic chamber set to 40°C, 40% relative humidity (rh). A normal thermoregulatory response results in the return of the soldier to training. If the thermoregulatory response is abnormal (~ 5% of cases), the soldier is scheduled for a second test 2 to 4 weeks later. If this test is again abnormal according to our standards, he is regarded as heat intolerant, and cannot continue his service in a combat unit. It is noteworthy that we would rather occasionally discharge a soldier from combat duty unnecessarily than have a heat-intolerant soldier return to it. (We do not advocate testing for malignant hyperthermia because the etiologies of malignant hyperthermia and EHS are different.)

Potential recruits to military service undergo the HTT as part of their medical evaluation if heat susceptibility is suspected based on family or medical history (eg, previous heatstroke, ectodermal dysplasia).

A QUESTION OF DIAGNOSIS

Theoretically, the diagnosis of EHS is simple: in a previously healthy individual who collapses while exerting in a hot environment for long periods and whose rectal temperature is above 40.5°C,[5–7] the diagnosis of heatstroke is virtually clinched. However, heatstroke is misdiagnosed when medical personnel adhere too rigidly to simplistic diagnostic schemes that require warm climate, very high body temperature, and lack of sweating.[5,7] It follows that the correct diagnosis of heatstroke should depend on the understanding of the composite clinical and pathological picture. The following case study is illustrative:

> Four days after recruitment, a healthy, 18-year-old military recruit participated in a 4-km march (average speed 4 km/h). The march started at 20:00 hours under a moderate heat load; the ambient temperature was 26°C, the rh, 78%. During the march he volunteered to carry a 10-L water canteen in addition to the 20-kg backpack that was carried by every soldier. On arrival at the base gate, the soldier collapsed. His face was red and he sweated excessively. At the base clinic, the patient was delirious, alternating with aggressive reaction, and he vomited several times. Body temperature was not measured.
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> Hyperventilation, aggressive reaction, the history of a previously healthy young man, and the short distance of march led the physician to the misdiagnosis of conversion reaction. He was wetted with about 40 L of tap water. About an hour after collapse, 10 mg diazepam was administered intramuscularly and another dose of 10 mg was administered intramuscularly 1 hour later. The patient's condition continued to deteriorate and convulsions, accompanied by dark-colored vomit, appeared. At 00:30 hours he was evacuated to a medical center.
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> On admission 4 hours after collapse, the patient was comatose, heart rate was 150 beats per minute, systolic blood pressure was 60 mm Hg, and breathing was shallow at a rate of 24 per minute. Rectal temperature (the first time measured!) was 39.6°C. Neurological examination revealed coma; spontaneous hand movements; low muscle tone; "doll eye" movement; dilated pupils, which reacted sluggishly to light; and diminished tendon reflexes.
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> Laboratory values for the arterial blood gas analysis were as follows: pH, 7.32; P_{O_2}, 58 mm Hg; P_{CO_2}, 30 mm Hg; and base excess, –9.5. Chemical and hematological findings were as follows: creatinine, 4.4 mg/dL; glucose, 20 (and later, 426 and 546) mg/dL; aspartate serum aminotransferase, 550 IU/L; alkaline

phosphatase, 114 IU/L; amylase, 165 IU/L; hemoglobin, 8.4 g/dL; hematocrit, 38%; white blood cell count, 16,000/mm^3 with neutrophilia and many immature forms; platelets, 55,000/mm^3; prothrombin time, 10% of normal; partial thromboplastin time, 85 seconds; and blood specimen, did not clot.

The patient suffered severe watery diarrhea. Urethral catheterization produced a small amount of residual urine, and during the next hours the patient remained comatose and totally anuric. Syringe needle punctures resulted in long-lasting bleeding, and spontaneous mouth and nose bleeding appeared. Dark material ("coffee grounds") was aspirated from the duodenal tube. The patient was infused with 13 L of crystalloid fluid, albumin, blood, fresh frozen plasma, and cryoprecipitate. Dopamine and steroids were administered. However, blood pressure remained low, bleeding diathesis did not abate, and bloody diarrhea appeared. The patient died 27 hours after the collapse.

Autopsy was performed several hours later. The body and brain were edematous and subcutaneous petechiae were seen all over the skin. Lungs were congested with interstitial hemorrhages. Small gas bubbles were noted in the heart. Liver was necrotic with massive bleeding. Hemorrhages were seen all along the intestinal wall. Multiple foci of hemorrhage were seen in the kidneys and the urinary bladder. A remarkable amount of blood was aspirated from the retroperitoneal space. Autopsy confirmed the diagnoses of heatstroke, disseminated intravascular coagulopathy (DIC), acute renal failure, and shock.

The performance of strenuous physical exercise (athletic events, military training, hard labor) in the heat has been notorious as the cause for heatstroke. However, in many cases heatstroke occurs also at relatively low ambient temperature.[8–11] Proper measurement of body temperature is essential, and hyperthermia should be expected. It should be acknowledged, however, that adherence to a diagnostic yardstick requiring that rectal temperature be high has proven to be inadequate and misleading. Although body temperature probably exceeds a critical temperature at the moment of collapse, in many instances lower temperatures are recorded; the first recordings of body temperature may be delayed, carried out by untrained individuals, or measured incorrectly.[4,12–14]

Contrary to earlier beliefs,[6,7,15] in most cases sweat glands are still active at the stage of heatstroke collapse, and profuse sweating is likely to be present.[13] Dry skin (*a*) is evident in situations where climate is very dry and sweat evaporates very easily or (*b*) is a late phenomenon of heatstroke, which usually coincides with a severe degree of dehydration.[16]

The loss of consciousness is a constant feature of heatstroke. This abruptly puts to an end the physical effort. Once activity ceases, body temperature falls and the victim will usually then spontaneously regain consciousness. Heatstroke patients are thus likely to present at the emergency ward with only mild hyperthermia and mild central nervous system (CNS) disturbances.

THE CLINICAL AND PATHOLOGICAL PICTURES

A typical presentation of heatstroke is the sudden collapse of a highly motivated, relatively untrained subject during physical exertion carried out in a warm climate (see Exhibit 8-1). The collapse is accompanied by loss of consciousness, very elevated body temperature, rapid pulse, tachypnea, hypotension, and shock. Nevertheless, this "optimal" scenario does not always occur. When an individual collapses under circumstances of physical exertion, the working diagnosis should be heatstroke, unless another cause is obvious.[4,5,17]

The early clinical signs of heatstroke are nonspecific. Therefore, any systemic disease or condition that appears with fever and manifestations of brain dysfunction must always be kept in mind but considered only after the diagnosis of heatstroke has been excluded (Exhibit 8-2).[4,18]

Prodromal Symptoms

The presentation of heatstroke is usually abrupt, but about 20% to 25% of all casualties have prodromal symptoms lasting minutes to hours.[8,19] These include dizziness, weakness, nausea, confusion, disorientation, drowsiness, and irrational behavior. Lack of recognition of the first signs of disability, and in some cases an assumption that the victim is malingering, have led to misassessment of the true physiological status.

Clinical Picture

The clinical manifestation of EHS reflects the result of a direct thermal injury and cardiovascular collapse. The high temperature will precipitate cellular biochemical disturbances, and CNS and secondary noncardiovascular changes. Usually a very distinct pattern of events, which can be categorized into three phases, is evident: the hyperthermic phase, the hematological and enzymatic phase, and the renal and hepatic phase.

EXHIBIT 8-2

DIFFERENTIAL DIAGNOSIS OF HEATSTROKE

- Severe dehydration
- Encephalitis, meningitis
- Coagulopathies
- Cerebrovascular accident, epilepsy
- Hypothalamic hemorrhage
- Hypoglycemia
- Drug intoxication
- Envenomization (eg, a bite or sting from a snake or bee)
- Allergic reaction, anaphylactic shock

Adapted with permission from Shapiro Y, Seidman DS. Field and clinical observations of exertional heat stroke patients. *Med Sci Sports Exerc.* 1990;22:8.

Hyperthermic Phase

CNS disturbances are present in all cases of heatstroke, as the brain is extremely sensitive to hyperthermia. Signs of depression of the CNS often appear simultaneously in the form of coma, stupor, or delirium, irritability, and aggressiveness.[5,20–22] Persisting coma after returning to normothermia is a poor prognostic sign.[5,19] Seizures occur in approximately 60% to 70% of cases.[13,19] Other findings include fecal incontinence, flaccidity, and hemiplegia.[5] Cerebellar symptoms, including ataxia and dysarthria, are prominent and may persist.[5,23,24] In more than two thirds of cases, pupils are constricted to pinpoint size.[19,25] Papilledema is present in cases of cerebral edema.[5] The cerebrospinal fluid and pressure are usually normal.[19,20]

CNS dysfunction is usually directly related to the duration of the hyperthermic phase and to circulatory failure. In most cases, coma persisting for as long as 24 hours, with subsequent seizures, may be followed by full recovery, without evidence of mental or neurological impairment.[20,26] Chronic disability may prevail for several weeks or months in the form of cerebellar deficits, hemiparesis, aphasia, and mental deficiency.[12,26,27] Only in rare cases, usually related to coma persisting for more than 24 hours, mental and neurological impairment may be chronic and persist for years.[28]

Gastrointestinal dysfunction, including diarrhea and vomiting, is a common occurrence. This may reflect poor perfusion or CNS impairment.[5]

Hyperventilation and elevation in body temperature are associated with primary respiratory alkalosis, which in cases of EHS, is masked by metabolic acidosis as a result of increased glycolysis and the development of hyperlactemia.[17,29–33] Hypoxia may be present in cases of respiratory complications.[6,29,34]

The Hematological and Enzymatic Phase

Heatstroke is associated with leukocytosis and significant alteration in absolute number and percentage of circulating lymphocyte subpopulations.[35] The white cell count may be in the range of 20,000/mm^3 to 30,000/mm^3 or even higher.[8,36] Hemorrhagic diathesis is frequently observed. It is clinically manifested by purpura, conjunctival hemorrhage, melena, bloody diarrhea, signs of hemolysis, hematuria, and pulmonary or myocardial hemorrhage.[5,22,37] The etiology is complex and multifactorial. It may be related to direct thermal effect on clotting factors (V, VIII),[38] decreased production of clotting factors due to hepatic dysfunction,[5,39] and thermal damage to the megakaryocytes in the bone marrow.[5,37] The most important mechanism may be a result of endothelial damage that leads to the release of thromboplastic substances, with resultant intravascular thrombosis and secondary fibrinolysis.[38,40] Hypofibrinogenemia, prolonged prothrombin time and partial thromboplastin time, elevated fibrin split products, and thrombocytopenia indicate the presence of DIC.[8,13,41,42] Clotting dysfunction peaks at 18 to 36 hours after the acute phase of heatstroke,[38] with prothrombin levels that reach a nadir on the second to third day.

One of the characteristic and almost pathognomonic features of EHS is the exceptionally high levels of various cellular enzymes. Evidence of skeletal muscle damage, displayed by elevation of serum creatine kinase (CK) activity (which is not always associated with myoglobinuria), occurs in a large percentage of patients with EHI. CK activity in the range of 10^3 to 10^4 IU/L is common (~ 5% of total CK may be of the myocardial isozyme, or MB, fraction [normal: 0–7 μg/L][12]). Peak values of CK occur 24 to 48 hours after collapse.[43] Although the dynamics in CK levels are not pathognomonic, they are helpful in making the differential diagnosis of heat-

stroke (see Exhibit 8-2) because in most other febrile states the enzyme levels will be within normal range. Elevated levels of serum aspartate aminotransferase (> 35 U/L), serum alanine aminotransferase (> 35 U/L), lactate dehydrogenase (> 190 U/L), and bilirubin (> 22 μmol/L) are also consistent features of heatstroke.[13,44] Levels of alanine aminotransferase greater than 1,000 U/L (normal: 10–35 U/L) are common in severe cases.[13,30]

Hypokalemia is found in the early stages of heatstroke.[5,32,45,46] Hyperkalemia may later develop as a result of metabolic acidosis, rhabdomyolysis, general cellular damage, and decreased renal perfusion.[12,13] Sodium levels are usually normal or slightly elevated due to dehydration.[47] In some cases, however, sodium levels may be slightly low, reflecting a state of hyperhydration (mainly because of unbalanced fluid replenishment during early treatment), or related to rhabdomyolysis.[48,49] Hypophosphatemia is frequently described as a result of the respiratory alkalosis[31,50] but has been observed also in patients with heatstroke who exhibit metabolic acidosis.[34,50,51] Hypocalcemia may reflect calcium phosphate or calcium carbonate deposition in damaged muscle.[12] In cases of acute oliguric renal failure associated with severe rhabdomyolysis, hypercalcemia has also been reported.[52] Hypomagnesemia may occur as a result of the increased loss of both calcium and magnesium in urine and sweat.[47]

Renal and Hepatic Phase

Disturbance in renal and hepatic functions characterize this late phase. High bilirubin levels, which may last several days, may reflect both hepatic dysfunction and hemolysis. Aminotransferases may originate from liver and muscle tissues.

Acute renal failure is a common complication of severe cases of EHS, occurring in approximately 25% to 30% of patients with EHS.[12,13,22] The etiology of acute renal failure is complex.[53] Decreased renal blood flow secondary to hypotension, following hypohydration and peripheral vasodilatation, is the major cause. Direct thermal injury may lead to widespread kidney tissue damage.[54] Intravascular clotting due to DIC may further contribute to acute oliguric renal failure.[40,54] Additional renal damage may also be inflicted by myoglobinuria.[55,56] Oliguria and anuria are characteristic features, the color of the urine being described as "machine oil," and with a low specific gravity.[13,53–55] Red and white blood cells, hyaline and granular casts, and mild to moderate proteinuria are commonly present.[54,55]

Pathological Picture

Degenerative changes and hemorrhages characterize the pathological picture. The former consist of swelling and degeneration of tissue and cell structures; the latter is often presented by widespread hemorrhages, which may vary in size from microscopic to massive bleeding.[57] The organs are generally described as congested or edematous, with increased weights and swollen cells.[5,8,13,22]

Brain edema or congestion is generally present. Swelling of nerve cells and degenerative neural changes are constant findings and could be traced through the successive stages from acute and chronic cell alteration to disappearance of neurons and their replacement by glia. Petechiae are common in the walls of the third ventricle and in the floor of the fourth ventricle.

Subendocardial hemorrhages, with predilection for the left side of the ventricular septum, are common, as is focal necrosis of the muscle fibers. Widespread necrosis of heart muscle, with a bleeding diathesis and myocardial infarction, are also described, with no abnormalities in coronary arteries. Striated muscles display degeneration, necrosis, and disruption of muscle fibers more prominently than in the myocardium. The lungs are congested and edematous. Numerous small pulmonary hemorrhages and massive pleural and intrapulmonary bleeding are presented. Pneumonia is occasionally evident.

The kidneys are always congested, with above-normal weights, and macroscopic hemorrhages are present in 20% of cases. Parenchymal damage is scant in very severe cases with early death. Pigmented casts, degeneration, and necrosis are often present in the lower nephron.

In the liver, centrolobular necrosis and hepatocellular degeneration are present around the widened centrolobular veins. Serial liver biopsies reveal hydropic swelling, extensive cholestasis, and leukocytic cholangitis.

Engorged intestinal vessels are a common finding, and the gastrointestinal tract may be the site of massive hemorrhages and ulcerations.

Subcutaneous petechiae are seen all over the skin. Degenerative changes are found in megakaryocytes, and basal cells and endothelial cell damage can be demonstrated. In small blood vessels, intravascular coagulation with thrombi is present.

MANAGEMENT AND PROGNOSIS

Heatstroke is the most serious of the syndromes associated with excess body heat.[58] It is defined as a condition in which body temperature is elevated to such a level that it becomes a noxious agent, causing damage to the body's tissues and giving rise to characteristic clinical and pathological syndromes affecting multiple organs.[13] The severity of the illness depends on (1) the degree of hyperthermia and (2) its duration, and is related to the duration of the temperature above a critical temperature.[59] Heatstroke is considered an extreme medical emergency that might result in death if not properly diagnosed and treated.

Management

To prevent or minimize expected complications, cooling should be initiated energetically immediately after the victim of EHS collapses, and minimally delayed only for vital resuscitation measures. In the field, the victim should be placed in the shade and his or her restrictive clothing removed. The skin must be kept wet with large quantities of tap water, and the body constantly fanned. These measures should not delay the rapid evacuation of the heatstroke victim to the nearest medical facility, which is of utmost importance.

Any time-consuming diagnostic procedures should be postponed until body temperature is controlled. At a medical facility, rapid cooling can be achieved by the use of ice packs, ice water baths, alcohol sponge baths, and with fans or air-conditioned rooms.[60–62] Sophisticated and expensive equipment, as suggested by Weiner and Khogali,[63] is unnecessary. The most practical and efficient method of cooling the body is by drenching it in large quantities of tap water. Tap water is readily available and does not require any complicated logistical arrangements. It eliminates the hazard of cold-induced vasoconstriction, which reduces the efficiency of heat dissipation.[61]

In heatstroke, the high body temperature is neither a fever nor the result of a hypermetabolic reaction but is rather the result of excess heat load (metabolic and environmental). Therefore, no drug is effective in reducing body temperature. Antipyretics should not be considered, as there is no shift in the thermoregulatory set point. Dantrolene is not effective because EHS is not linked to the uncontrolled release of calcium ions from the sarcoplasmic reticulum.

Rectal temperature must be measured to confirm the diagnosis of EHS. It is noteworthy, however, that in many instances the initial measurement will be lower than the critical core temperature (~ 41.0°C). This is due mostly to a delay in getting the first measurement, or due to faulty measurement.[4,12–14]

Drastic drop in skin temperature may induce violent shivering. Chlorpromazine (50 mg administered intravenously[18]) or diazepam[60] have been found to be effective in depressing the shivering and preventing an increase in metabolic heat production.

Blood pressure and pulse must be checked, a quick clinical examination performed, and if possible, urine and blood should be obtained for examination, prior to infusion of fluids.

Electrocardiographic findings are nonspecific and may include ST segment and T wave abnormalities and conductive disturbances. However, the evidence for such abnormalities in young patients with EHS is scarce. Nevertheless, continuous cardiac monitoring is recommended during the first 24 hours of hospitalization because of the potential rhythm disturbances that may emerge from electrolyte and acid–base imbalance.

The airway must be kept clear, and if the patient is comatose, insertion of a cuffed endotracheal tube should be considered because aspiration is common, especially when seizures occur.[4] If available, oxygen administration is advisable as metabolic demands are high and oxygenation may be hampered by pulmonary complications.[6] Positive pressure ventilation is indicated when supplemental oxygen alone does not suffice.[4,64]

A large-bore (eg, 18 or 20 French) intravenous catheter should be inserted without delay. When seizures occur they add to body heat storage, and thus cooling is inefficient; repeated doses of diazepam (5–10 mg) should be administered intravenously until the convulsions are controlled. Two liters of Ringer's lactate or saline should be infused in the first hour; later, fluids should be administered in accordance with the state of hydration.

Depending on the patient's hydration state, continuous fluid monitoring is required during the first 24 hours of hospitalization. A central venous catheter may be required to identify hypovolemic patients. A Swan-Ganz pulmonary artery catheter can provide more accurate guidance to fluid therapy when the circulatory state of the patient is unclear.[60] Monitoring the urinary flow using an indwelling urinary bladder catheter provides important information on the patient's state of hydration. Furthermore, urinary output reflects the kidneys' perfusion level and allows an early diagnosis of acute oliguric renal failure. Intravenous mannitol (0.25 mg/kg) or intravenous

furosemide (1 mg/kg) may be used to promote diuresis and prevent damage from myoglobinuria and hyperuricemia.[6,60] Anuria, uremia, and hyperkalemia are indications for peritoneal dialysis or hemodialysis.[65]

Acid–base abnormalities are usually corrected by cooling and by proper hydration not requiring specific treatment. Serum electrolyte imbalances should be followed closely, especially with regard to their relation with possible cardiac arrhythmias. Glucose levels should be checked repeatedly, as both hypoglycemia and hyperglycemia have been reported following heatstroke.[47]

Coagulation tests must always be performed on admission and at 12-hour intervals afterward. DIC may be recognized only after 24 to 72 hours. It starts abruptly and may be severe. Bleeding from venipuncture sites is characteristic; other common sites are bleeding into the skin, from the nose, gums, and respiratory and gastrointestinal tracts. The deficiency in clotting factors should be corrected by transfusion of fresh frozen plasma, cryoprecipitate, or platelet concentrates.

Acute hepatic dysfunction is exhibited by elevated levels of transaminases and bilirubin; peak levels are seen 36 to 72 hours after collapse, and high levels may last for several days.[13,43] Muscle damage is displayed primarily by marked elevation of serum CK activity levels, which peak 24 to 48 hours after collapse and usually recover spontaneously within 5 days.[43] Muscle and liver enzymes and bilirubin should be monitored but usually no drastic intervention (such as liver transplant) is necessary or advocated.

Prognosis

Since the end of the 1960s, it has been shown that more than 95% of victims of EHS survive when the following four measures are implemented[17,25]:

1. rapid decrease of body temperature,
2. arrest of convulsions,
3. proper rehydration at the site of the event, and
4. quick evacuation to a medical center.

The noxious effect on the tissues caused by heatstroke is directly and closely correlated with the degree of hyperthermia and its duration, and is a function of both the *elevation* and the *duration* of the elevation of the temperature above the critical value (the integrated time–temperature area).[59] Predictors of poor prognosis include (1) body temperature above 42°C,[13,25] (2) prolonged duration of hyperpyrexia, (3) prolonged coma, (4) hyperkalemia, (5) oliguric renal failure, and (6) high levels of aminotransferases.[17] The vast majority of survivors from EHS recover without sequelae.[6,13,26] However, some neurological deficits may be apparent in some patients, usually for only 12 to 24 months and only in rare cases for longer.[28]

According to the IDF experience, only six fatal cases of EHS were recorded in soldiers since 1980; in all cases, misdiagnosis, ineffective primary treatment, and delay in evacuation were the major causes for deterioration of the casualty's condition.

CONCLUSION

EHS is a state of extreme hyperthermia that occurs when heat that is generated by muscular exercise exceeds the body's ability to dissipate it at the same rate. The elevated body temperature becomes a noxious agent causing damage to the body's tissue, affecting multiple organs, and may be fatal if misdiagnosed or diagnosis is delayed. EHS is underdiagnosed because of adherence to simplistic and misleading diagnostic schemes that require long exertions in very warm climates and with very high body temperatures (> 40°C). Prompt diagnosis and treatment result in full recovery of most patients. Primary treatment should focus on clearing the airway, measuring rectal temperature, cooling the whole body, controlling convulsions, and prompt evacuation to a hospital.

The Israeli experience has proven that EHS can, in most cases, be prevented, education being the mainstay of this prevention. By providing proper instructions and following some simple regulations, the incidence of EHS has been reduced markedly. These instructions and regulations include grading training programs and matching effort to the individual's capacity, limiting physical activity under severe heat load, scheduling proper work/rest cycles, and providing adequate rehydration routines. Also, regulations for monitoring the medical status of soldiers participating in physical activities and evaluating the soldiers' medical histories should be implemented.

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